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Case Report

Correction: An Uncommon Cause of MINOCA: Mad Honey Poisoning

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Abstract

Consumption of grayanotoxin-containing honey that is produced from flowers of the Rhododendron family causes mad honey intoxication, in other words grayanotoxin poisoning (1). This flower family grows in Japan, Nepal, Brazil, parts of North America, and the eastern Black Sea region of Turkey. Grayanotoxin is accountable for clinical presentation. Dose-dependent, grayanotoxin causes different clinical conditions from dizziness, hypotension, and bradycardia to impaired consciousness, syncope, atrioventricular block, and asystole due to vagal stimulation. (2). But consumption of mad honey is a rare cause of acute coronary syndrome. Herein, we present the case of a 49-year-old man who presented to hospital with dizziness whose electrocardiogram (ECG) shows sign of acute inferior myocardial infarction after consumption of mad honey.

Introduction

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Case Report

In May 2020, a 49-year-old man was admitted to the Emergency Department (ED) of Recep Tayyip Erdogan University Education and Research Hospital with dizziness, cold sweating and blurred vision. The patient provided written informed consent. There was no chest pain. He said that his complaints began after ingesting 2 tablespoons of mad honey. There was no medical history. He is a smoker for 30 pack-years. The patient's neurological examination was normal. His arterial blood pressure was 70/40 mmHg and heart rate 49 beats/min. The ECG showed ST elevation on DII-DIII-AVF derivations (inferior derivations) and ST depression in aVL derivation (Figure 1). Parenteral fluid infusion with 0.9% sodium chloride (NaCl) (approximately 2000 cc) and 1 mg atropine sulphate was administered. Routine biochemical test, hemogram and cardiac enzymes was checked. He was monitored closely. One hour later arterial blood pressure was 118/70 and heart rate 62 beats/min. Routine blood test including cardiac enzymes were at normal range. The patient's symptoms relieved during following-up at ED. But there were persistence ST elevation on the second ECG. The echocardiography was showed no wall motion abnormalities. Therefore, the patient was admitted to coronary intensive care unit to perform coronary angiography